Biological Uniqueness and the Quality of Life

RECENT ADVANCES in biological science, particularly in the field known as molecular biology, have demonstrated beyond any doubt the biochemical and therefore the biological uniqueness of every person. Each human life starts with a unique set of genes whose chemical structure determines the physical, mental and emotional endowment of the person. And then, from the moment of conception and until death, the environmental experience which is also unique for each person reacts with this body chemistry to influence, modify and change it. Thus every person, even each of genetically identical twins, is biochemically and biologically unique with unique needs for personal fulfillment to give quality to his or her life.

So far, biological science has provided little hard data to explain the origin of life and even less upon its value or purpose. Nor does biological science say anything about the meaning or implication of biological uniqueness in terms of the human potential for personal fulfillment or the quality of the life which is to be led. It is here that biological science merges into medicine and medicine merges into the art and science of living within biological law and the physical and cultural framework of the world biosphere and of modern human society.

What are the implications of this for medicine? First it suggests a core tenet for medicine. Not only practicing physicians but the profession as a whole should give greater recognition to the importance of the fundamental uniqueness of each person, and all that this in turn implies for patient care and indeed for social, economic, political and even legislative action by the profession. And second it suggests that personal fulfillment and the quality of life will also be different and unique for each person, and that this will be what is attainable within his or her physical, emotional and situational capabilities and limitations. In terms of human society and the world biosphere, personal fulfillment must of course be tempered by the rights and needs of others for the same, and by the need to create and preserve an environment which will support the quality of life which is desired.

The particular professional expertise of medicine lies in illness and health. However, these are not to be separated from the reality of biological individuality, the goal of personal fulfillment, the quality of life, and the environmental situation which all pertain uniquely to every human being. They are each part and parcel of the diagnosis and treatment of illness and of the restoration and maintenance of health. The implications of this for the practice of medicine are enormous and extend far beyond patient care and into the role of medicine in the social, economic, political and legislative framework of the world biosphere and modern society. This has yet to be explored.

It is suggested that there is great potential for prestige and power in this for modern medicine in modern society. A posture of strong advocacy of the fact of biological uniqueness of every person which links the goal of personal fulfillment and the quality of life to health, illness, and social, economic and political progress, would surely be worthy of our profession. If properly developed and properly applied, such a posture could become a tremendous force for "the betterment of the public health" in our evolving society.

—MSMW

Treatment of Ventricular Septal Defect

IN THE SPECIALTY CONFERENCE which appears elsewhere in this issue of THE WESTERN JOURNAL OF MEDICINE, Dr. Friedman discusses thoroughly a common, major congenital heart lesion—the large ventricular septal defect causing congestive heart failure and retarded growth. Because of the importance of the subject some added emphasis and qualifications are set down here.

The isotope dilution technique described is not in general use. It is semi-quantitative at best, not able to detect very small shunts, does not define the site of the defect and, being empirical, is subject to errors due to unknown variables. It may be replaced by the more soundly based gamma function analysis recently described and is better used for screening and follow-up than for diagnosis.

The data of Charts 1 and 2 in the Specialty

Conference might be clearer if the natural histories of small, moderate and large defects are separated.

Small defects (murmur, no or minimal cardiomegaly, normal pulmonary arterial pressures, pulmonary blood flows under twice systemic flows) are asymptomatic and they make up 65 to 75 percent of all ventricular septal defects at birth. They do not cause heart failure or pulmonary vascular disease, and the major complication associated with them—bacterial endocarditis—is probably uncommon. About 50 to 75 percent of them close spontaneously by 15 years of age, and Dr. Friedman emphasizes correctly that the risk of treating small defects conservatively is less than the risk of surgical complications of defect closure.

Moderate defects (moderate cardiomegaly, pulmonic flow two to three times systemic flow, pulmonary arterial pressure under 50 percent of systemic pressure) usually cause moderate congestive heart failure that responds well to medical treatment. These defects account for 20 to 25 percent of ventricular septal defects at birth. They tend to close or get smaller spontaneously, usually under three years of age but sometimes after many decades. If they remain moderate sized, pulmonary vascular disease may develop after 15 years of age, as may occur in physiologically similar atrial septal defects. Therefore we recommend surgical closure at about five years of age when there are big shunts and big hearts or symptoms. Smaller defects in this group can be treated conservatively.

The large defects account for 10 to 15 percent of ventricular septal defects at birth. As Dr. Friedman mentioned, the left-to-right shunt increases as the high fetal pulmonary vascular resistance falls after birth. In term infants the increasing left ventricular volume load usually causes overt congestive heart failure between six weeks and six months of age, but in preterm infants, with a lower pulmonary vascular resistance at birth, heart failure with a big defect usually presents before two months of age. At any age, overt congestive heart failure is usually preceded by irritability, excessive sweating, poor weight gain and tachypnea.

Rarely, when heart failure is intractable, early operation is needed. In many centers, surgical closure of the defect has replaced pulmonary artery banding. Banding is often imperfect, has a 6 to 10 percent mortality rate, has complications (for example, obstruction of one pulmonary artery, thickening of the pulmonary valve) and increases the duration and mortality of subsequent corrective surgery. If a surgical team can close defects in infants with a mortality rate of less than 10 percent, banding should be restricted to unusual problems such as multiple muscular defects and associated complicating lesions.

If congestive heart failure is partly controlled but growth is poor, many cardiologists would disagree with Dr. Friedman that early operation is warranted, especially if this means an extra operation (banding) or the higher risk of closing the defect in a tiny infant. Our group would defer operation to allow the child to grow and improvement to come about spontaneously. Spontaneous closure occurs in 5 to 10 percent of the large defects and as many as 50 percent of them may get small enough to eliminate concern. If the defect remains large, however, then the risk of obliterative pulmonary vascular disease is high. Both a smaller defect and pulmonary vascular disease will decrease the shunt and cause subjective and objective improvement.

Unfortunately, cardiac catheterization is the only certain way of determining if pulmonary vascular disease is beginning. The pulmonic component of the second heart sound is loud in large ventricular septal defects whether resistance is high or low, and by the time the smaller heart as observed on x-ray films and pure right ventricular hypertrophy as observed by electrocardiogram indicate pronounced pulmonary vascular disease, it may be irreversible. Therefore the safest way of handling these infants is to do cardiac catheterization studies when they present with heart failure (to define anatomy and physiology) and then repeat such studies six to nine months later to determine changes in defect size and pulmonary vascular resistance. If a rising resistance is found, it is usually reversible after closure of the defect, an operation that can be safely undertaken at this age. Research into non-invasive methods of measuring pulmonary blood flow may allow better timing of recatheterization in future, but even at present in experienced laboratories cardiac catheterization is safe enough to be used, more than once if necessary, to select the appropriate patient and time for corrective operation.

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